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COMPLIMENTS OF
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LIMITATIONS
OF THE
VISUAL FIELD OF INTRACRANIAL ORIGIN.

BY

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Surgeon Oculist and Aurist to the Montreal Dispensary and to the Foundling Hospital.

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SAINT SULPICE

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LIMITATIONS
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VISUAL FIELD OF INTRACRANIAL ORIGIN.[1]

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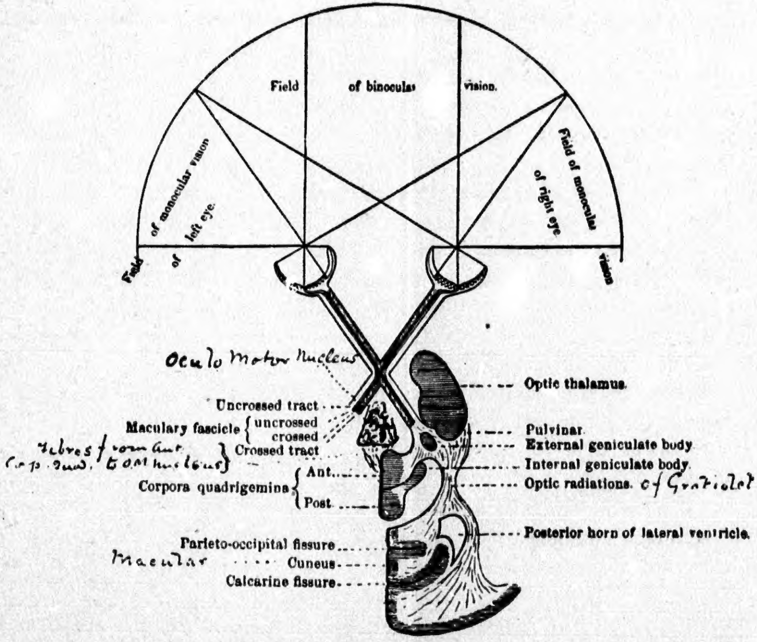
In reading this paper to you this evening, I do not offer you anything very new or original, but simply will give a *précis* of some interesting cases and the presumed or defined lesion in the brain which caused the limitation of their visual field.

I will take the liberty before going into the subject matter proper, of drawing your ettention briefly to the present status of our knowledge as to the anatomy, both gross and minute, of the visual tracts and their central connections in the brain

FIG. 5


Field of binocular vision.
Field of monocular vision of left eye
Field of monocular vision of right eye
Oculo Motor Nucleus
Uncrossed tract
Macular fascicle { uncrossed crossed
Fibres from Ant. Corp. Quad. to OM nucleus
Crossed tract
Corpora quadrigemina { Ant. Post.
Parieto-occipital fissure
Macular
Cuneus
Calcarine fissure
Optic thalamus.
Pulvinar.
External geniculate body.
Internal geniculate body.
Optic radiations. of Gratiolet
Posterior horn of lateral ventricle.

The fields of vision are, as you know, divided into an inner and

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outer, or nasal and temporal portions—the dividing line being fairly sharply defined and passing vertically through the fixation point. The nerve fibres from the retina are united in the optic nerve as far as the chiasma, where the fibres from the nasal portion of the retina decussate with the corresponding fibres from the opposite eye.

Before following them farther, it is as well to draw attention to the fact that, as a general rule, the field for a short distance on each side of the line dividing the nasal and temporal portions of it, is innervated with both sets of fibres, so that in a case of hemianopsia, the blind area limit would be clear of the fixation point.

From the chiasma the fibres from the corresponding halves of the retina pass through the optic tract to the basal ophthalmic ganglia, viz., the pulvinar of the optic thalamus, the external geniculate body and the anterior corpus quadrigeminum.

From the anterior corpus quadrigeminum fibres run to the nuclei of the ocular muscles—others decussate with fibres from the opposite corpus quadrigeminum in the roof of the aqueduct of Sylvius and thence pass to the fillet which runs on in the tegmentum of the cerebral peduncle to the optic thalamus.

The fillet contains the sensory fibres from the opposite half of the body.

From the arm of the anterior corpus quadrigeminum fibres go to the corona radiata and so on to the occipital cortex in the region of the cuneus where the centres for vision lie.

From the pulvinar and external geniculate bodies, fibres enter Gratiolets optic radiations (or corona radiata) in the posterior third of the internal capsule between the lenticular nucleus and optic thalamus and thus run on to the occipital cortex.

The external geniculate bodies receive mainly the fibres from the macula of each eye, according to Knies.

In the chiasma in addition to the optic nerve fibres are two bundles of fibres lying posteriorly and superiorly called respectively Guddens and Meynerts Commissures.

Gudden's commissure connects both posterior corpora quadrigemina and internal geniculate bodies and sends fibres directly into the corona radiata of the occipital lobe on the same side.

This commissure seems to have no connection with vision, but rather to act as an auditory chiasm.

Each convolution of the cerebrum is connected with adjacent convolutions by association fibres, as is also each lobe of the same hemisphere—and also homonymous parts of each hemisphere by commissural fibres. Hence the different portious of the brain are intimately connected.

Again the centrifugal and centripetal connection of the cortex with the rest of the brain is by the corona radiata in which the fibres are collected in the internal capsule.

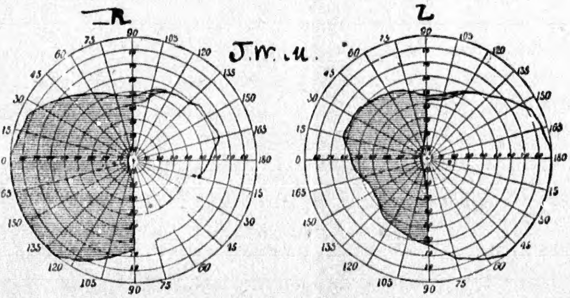
Of the association fibres, one of the most important bundles is that connecting the visual cortex with Broca's frontal convolution, on account of the latter's relation to speech.

A point that Knies brings out is the presence of centripetal and centrifugal fibres in the optic nerve, the former being much finer than the latter and originating in the ganglion layer of the retina, to terminate in a fine plexus in the optic basal ganglia. The centrifugal originate in the ganglion cells of the three basal optic nuclei, are coarser and come to an end in the internal granular layer of the retina.

As to the connection of the optic tracts with the pupillary nucleus, it is a question whether the fibres from the tracts pass through the anterior corpora quadrigemina or external geniculate body and pulvinar or whether they pass from the tract close to these bodies directly to the ganglion habenulæ.

A word as to the location of the cortical visual centres. The macula portion of the field corresponds to the cuneus and first occipital convolution. Of the rest, of the occipital cortex, the anterior portion corresponds to the lower portion of the field of vision, the posterior to the upper portion, and the lateral to the outer portion.

The first case I will mention is, J. W. McK., aged 29. I saw him in October, 1895. Nine months previously, he was troubled with severe headaches and vomiting. Three months later he had an attack of Jacksonian epilepsy for the first time, which after this frequently recurred.



In these attacks the left arm was drawn up over the head, the left leg was drawn up flexed, the head was drawn back and to the left and the eyes rolled up and to the left.

Vision had been failing latterly. His condition on October 7th, when I first saw him was as follows:—

Complete left hemiplegia.
Vision L. E., $\frac{5}{36}$.
" R. E., $\frac{5}{9}$.

Both pupils active to light and accommodation, also consensually, also on stimulating the blind retinal area.

There was left hemianopsia, the dividing line clearing the fixation point. The right sides of both fields were slightly contracted, but the colour vision field, although contracted, was normal as to perception of colours.

In both fundi one saw œdematous choked discs, the surrounding retina being only slightly swollen, and there being no hæmorrhages. The visible disease was most advanced in the left papilla. Mentition and speech were slow. The patient died six weeks later, a post-mortem was performed by the local medical man in the country, and the report was not very explicit, being merely that he found a tumour in the motor area of the right cortex and neighbourhood.

The case presents a most interesting clinical picture.

The irritative symptoms of the Jacksonian epilepsy clearly would point to the seat of the lesion as being in the right motor area, yet this could not be absolutely affirmed, since the distant symptoms of a cerebral tumour are so varied.

The later development, however, of the marked paralytic symptoms on the opposite half the body pretty well removed any possible doubt. The further appearance of left hemianopsia with retained pupillary reflex would point to a lesion posterior to the basal optic nuclei, either in Gratiolet's radiations or the cerebral occipital cortex.

I may here mention that this pupillary reaction from stimulation of the blind area of the retina in these cases I never feel absolutely sure of, if the reflex is present—as the test is delicate—more certainty can be felt if it is absent; the latter pointing to a lesion at or in front of the basal optic nuclei.

The growth must also thus implicate the visual cortex on the right side directly either in the cortical centre or the radiations, likely the former.

It would hardly be in the radiations without causing other paralyses of ordinary sensation—hemianæsthesia.

The contracted fields on the right side must be ascribed to the choked disc condition, with the accompanying interference of conduction in the nerve—although the diminution in vision is far from being constantly present in choked disc.

The appearance of the discs was rather that of œdematous infiltration than an inflammatory process.

Knies explains this condition as due to increased intracranial pressure.

He says that normally there is an outflow from the eye towards the brain within both the nerve and its sheaths. Increased intracranial pressure would abolish this; stasis and œdema would develop.

This œdema becomes evident in the optic nerve outside the cranial cavity, where it is not enclosed in a tightly fitting sheath, i.e., at the disc, and also within the vaginal spaces where the external sheath is thinnest and yielding; immediately behind the eye.

Persistence and increase of this pressure on the nerve can in time obstruct vision. Now as to the movements of the eyes up and to the left, this symptom occurring early, during the Jacksonian epilepsy could be due to the irritation of the visual spheres, taking into account the later appearance of the left hemianopsia. Although one need not look so far afield as this, for it is well-known that conjugate deviation towards the opposite side can be called forth on irritation of almost all parts of the cerebral cortex.

Yet it is most surely done by stimulating the visual cerebral cortex, Munk showed that irritation of the anterior zone caused the eyes to be directed downwards and of the posterior zone to be directed upwards as well as to the opposite side.

Thus the clinical picture pointed very clearly to what we might expect to find at the post-mortem examination.

The next case I will describe to you also came to section so that the diagnosis could be verified; it has already been reported by Dr. James Stewart.

It is very interesting in this connection in that the lesion was situated in an entirely different portion of the visual tract from the last.

J. W., aged 42, came to me in February, 1888, with total blindness of the left eye and complete right hemianopsia of the right eye. The left eye had been failing since two years, and the right for eighteen months, but the latter had remained apparently stationary since eight months.

He had pains in joints and hands; there is some giddiness with a tendency to fall forward: no history of syphilis.

Has been a rather heavy drinker. Vision L. E., no perception of light, pupil semidilated immobile, but reacts on stimulating the R E. with light.

R. E., complete blindness to outer side of field, the inner side was amblyopic; vision being reduced to counting fingers at 12 ft. In reading type can pick out a letter of the largest size (No. 16) here and there. In this area the perception of green was very defective, also after looking at green for some time there was an after image in pink.

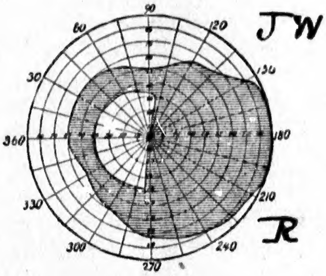
Examination of the fundus revealed, in the L. E. white atrophy of the disc, with vessels somewhat contracted.

R. E. disc not so pale as in left eye.

His reflexes were slightly exaggerated, but there were no motor disturbances.

Frequent flushings and perspiration of face and back of neck occurred.

The further course of this case was very slow, six years elapsing before his death.



His sight entirely failed about eighteen months after I saw him. He became fat and flabby, always had a good appetite, but was greatly bothered with continuous sleepiness.

Frequent flashings of light (Photopsiæ) appeared, and later on he developed hallucinations of "beasties" crawling over him, which he imagined he could point out. The post-mortem examination revealed a tumour as large as a hen's egg, springing from the meninges and pressing on the front of the chiasma.

The microscopic investigation showed the growth to be of the nature of an endothelioma.

The diagnosis made at the first time Dr. Stewart and I saw him was that of cerebral tumour, implicating the chiasma from the front.

The visual disturbance was the only factor to base the diagnosis on, and if one could only have seen the patient earlier the limitation of the field would have been more typical.

The absence of the pupillary reflex showed that the lesion must be somewhere between the left eye and the basal optic nuclei, also the presence of visible optic atrophy was, if necessary, a proof against the lesion being further back than the basal nuclei, as in lesions of the optic radiations or cortex, white essential atrophy is exceedingly rarely, if ever visible at the optic disc.

Again and mainly, it would be hardly possible to have a lesion causing bitemporal hemianopsia anywhere else than in the anterior part of the chiasma, unless we could imagine a lesion of each central nasal area. The portions of the optic nerve supplying the inner halves of the retina decussate in the anterior part of the chiasma, a further extension of the growth to the left would easily obliterate the function of the remaining portion of the left optic nerve causing complete blindness of that eye and subsequent atrophy.

When I first saw the patient the obtunding of the nasal half of the right field, together with the defective perception of green, pointed to marked interference with the conduction of the remaining active portion of this nerve; there was a concentric and fairly equal contraction of the field for white and colours pointing to a likely slow growth; if on the other hand the field for colours had been much more markedly contracted for colours than for white, the prognosis would be a rapid progress of the disease.

Another striking point was the absence of any optic neuritis or choked disc.

It is likely the very slow growth of the tumour would account for the absence of the choked disc, time being sufficient to allow of the formation of lateral drainage or gradual distension of the sheath.

The hallucinations point to the cortex being still functional (it being the seat of conscious vision) and to its irritation.

Another interesting case in which the visual disturbance was due, not directly to the intracranial lesion, but to the optic neuritis set up by it, is the following:

G. J., aged 12, was referred to me by Dr. Springle for examination of the ocular condition.

He had for some weeks been suffering from severe headaches, occasional vomiting and giddiness.

His vision was $\frac{5}{18}$ with each eye, but no lens could further improve it.

The pupils reacted to light but were sluggish, they were even.

The field was slightly contracted peripherally, and the perception of green uncertain.

The fundus showed marked inflammatory choked disc, with great swelling (3 mm.) extending well into the surrounding retina and numerous small hæmorrhages. The condition was if anything more marked in the right.

The boy complained of the headaches being general, and continuous with occasional exacerbations. He was markedly giddy, with a tendency to fall forward, a very unsteady reeling gait, knee reflexes exaggerated, but no other motor or sensory disturbance.

This case got rapidly worse, complete blindness ensued within four weeks, convulsions, general in character, set in which became almost continuous, and finally coma and death.

I am able to show you the brain. At the post-mortem examination we found almost the entire vermiform process of the cerebellum occupied by a tumour which had undergone softening at its centre; it was strictly limited to the vermiform process and the immediately adjoining cerebellum.

The main localizing symptom here was the vertigo. The cerebellar coordination and integration of the body movements being interfered with ; the direct cerebellar tracts which convey to the cerebellum, sensory impressions indicating the posture of the body, being partly destroyed. The inflammatory nature of the choked disc differed entirely from the œdematous form I mentioned as recurring in my first case of cerebral tumour.

According to Leber and others, tumours especially of the cerebellum and base, if rapidly growing, excrete irritating and inflammatory substances. These are diffused through the cerebro spinal fluid and enter the sheath of the optic nerve, where from increased intracranial pressure stasis has already existed and here where œdema has developed, the substances set up inflammatory action.

The narrowing of the field, together with the faulty perception of green is typical of interference with conduction in the nerve.

The diagnostic value of the double optic neuritis in cerebral tumours is well known.

The next case is one of hemianopsia and hemiplegia due, I think, to embolism or thrombosis.

T. H., aged 68, a diabetic, came to me last autumn for the examination of his eyes for glasses. He was suffering from right hemiplegia and stated that some months previously he had a paralytic stroke. After this he was for a short time aphasic but this symptom gradually disappeared.

On examining his eyes I found, in addition to some opacities of the lens, well marked right homonymous hemianopsia, and this was the main cause of his complaint, on account of the difficulty in reading, the inability to see words ahead on the line being very troublesome.

The pupils were even and active, on stimulating both the blind and normal portions of the field.

Seven months later the general condition was the same.

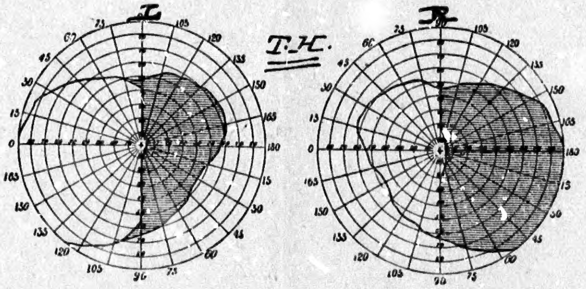
There was no hemianesthesia at any time.

There is some difficulty in locating this lesion as the areas affected are so diverse.

In the ocular symptoms the presence of the pupillary reaction point to the lesion being somewhere behind the basal optic nuclei on the left side, either in Gratiolet's optic radiations in the internal capsule or in the cortex.

It is unlikely to have been in the former as there was no anæsthesia, and the fibres of general sensation run in the same portion of the internal capsule—while still further in front are the motor fibres. Now these latter and the special sense optic fibres could hardly be affected in this region, while the fibres of ordinary sensation escaped. Hence the lesion must be cortical, and could hardly be otherwise than thrombotic or embolic in the middle cerebral artery.

The aphasia was a transitory, remote symptom.



According to Seguin, a lesion causing typical hemiplegia, aphasia if the right side is paralysed, little or no anæsthesia, and lateral hemianopsia is probably due to disease in the area supplied by the middle cerebral artery.

An exceedingly interesting and rather peculiar case of colour blindness in homonymus halves of the field of vision came under my observation some years since.

J. K., aged 35, suffering from incipient general paralysis of the insane was referred to me by Dr. James Stewart.

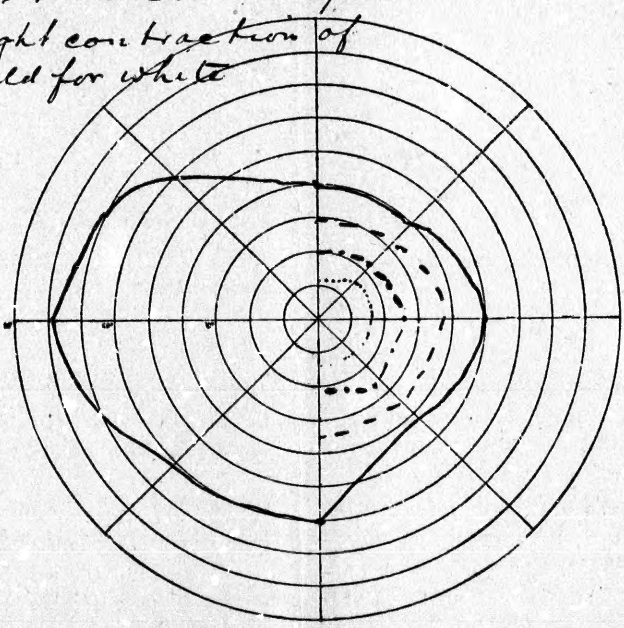
The vision was $\frac{5}{6}$, or approximately normal, the pupils even and active and the fields free for white but total colour blindness existed to the left side of both fields of vision.

He had left facial paresis. Beyond this and the mental condition there was nothing to note.

With the reaction of the pupils present, the condition was plainly referable to the right occipital cortex.

An interesting point was the retention of such good central vision.

Wilbrand brought forward the theory some years since that the elements for the perception of light, space and colour in the cortex were superimposed above one another in the above order from within outward, hence the colour sense could be interfered with, without the light or space sense being affected, and again the light elements could not be interfered with, without the space and colour elements also suffering. Accordingly hemiachromatopsia would be due to superficial disturbances of the cortex. This I mentioned some years ago in a paper I read in Ottawa.



J K
1889

L. E

Left Hemiachromatopsia
slight contraction of
field for white

As Knies, however, points out, anatomical proof of this is wanting and he brings forward a far more sensible assumption, that the function in the cortex is merely disturbed or rendered difficult, interference with conduction in the neuropilemma or the ganglion cells exhibit diminished function and capacity for action, which may be temporary or remain stationary.

Likely here we get a manifestation in this area of the general pathological state whatever it may be, which is associated with the general paralysis.

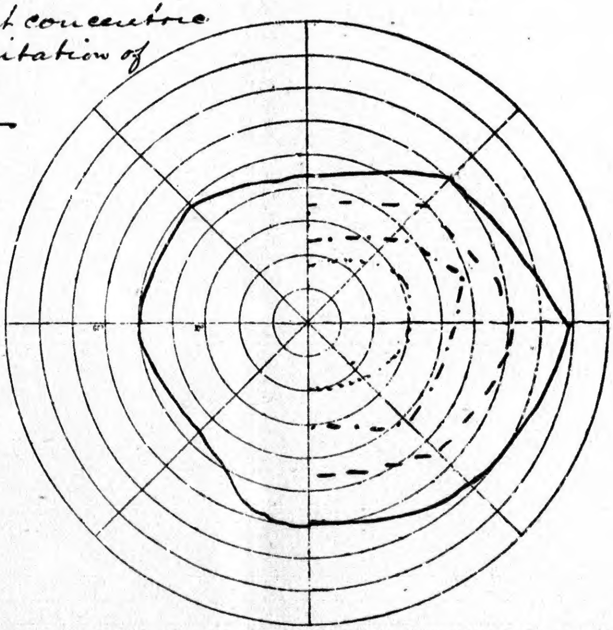
I have now brought before you a series of cases of visual disturbance due to brain lesions, each of them fairly characteristic, and happily in three cases, the post mortem reports were secured to complete them.

The first of left hemiplegia and hemianopsia, with a tumour in the right motor area and neighbourhood, producing œdematous choked disc

J K
1889

R. E.

Left
Hemiachromatopsia
Slight concentric
limitation of
field



The second of tumour of very slow growth in the chiasma area with typical field limitations and setting up pure atropy of the optic nerves.

The third case was the cerebellar tumour, setting up optic neuritis and consequent blindness.

The fourth was a case of hemianopsia and hemiplegia, due likely to thrombosis or embolism.

The fifth was the interesting case of hemiachromatopsia.

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